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Editorial: Cardiac Disease – What is Different in Patients with Renal Insufficiency

G. Mayer

There is a tremendous increase in the prevalence and incidence of end stage renal disease (ESRD) worldwide. According to the Austrian Dialysis and Transplantation Registry the number of incident patients on renal replacement therapy was approximately 50/year in 1970 but has increased to more than 1000/year since then. Even though remarkable technical and clinical progress has been made in dialysis and transplantation, the life expectancy of a patient with end stage renal disease is still reduced by as much as 50–75 % when compared to the general population. Even though the causes of death vary according to the mode of renal replacement treatment, cardiovascular disease is the major reason in dialysis patients (59 % in Austria) as well as after transplantation (54 %). This fact is in contrast to the general population as cardiovascular morbidity and mortality in ESRD patients is still increasing. Several attempts have been made to explain this finding. First it has to be considered that the age structure of the dialysis population is changing, with increasingly older patients (with more comorbid conditions) starting renal replacement therapy. In Austria, the number of prevalent patients within the age group up to 30 years has remained stable during the last 10 years, however, the number of patients aged 55 to 79 has almost doubled (accounting now for almost 50 % of the total population). Furthermore, as diabetes mellitus type II is now one of the leading reasons for ESRD, the number of comorbid conditions in incident dialysis patients also has increased. In 1993 in the United States 8.5 million patients left the hospital after an acute myocardial infarction or after stroke. This number increased to 9.7 mio. in 1993. Of these 1.2 million "excess" survivors, 1800 reached end stage renal disease within 3 years. Thus, as the number of incident dialysis patients increased by 9000 from 1990 to 1993, 20 % of this increase can be explained solely by better survival after major cardiovascular complications.

The situation is furthermore complicated by the fact that heart disease in patients with end stage renal failure differs in many aspects from one in the non-uraemic population.

We recently were able to show that the complex changes in the vessels and myocardium observed in renal patients complicate diagnosis [1]. Angina is much less sensitive and specific because "silent ischaemia" is common (perhaps due to neuropathy) and quite often typical chest pain is observed despite "normal" coronary arteries on angiography. Additionally, non-invasive screening tests such as treadmill exercise often cannot be reliably performed due to an inability of the patients to reach the level of exercise needed. At least in our hands pharmacological stress enhanced nuclear imaging techniques also have an extremely low sensitivity and specificity. Other authors have pointed out that, even stress echocardiography is a poor indicator in patients with terminal renal failure. When compared to the general population the pathogenesis of heart disease in ESRD subjects is also different. Even though coronary lesions (which may be progressing at a faster rate) account for some changes, alterations in myocardial structure and composition also contribute to the development of heart failure. As pointed out by Amann and Ritz in this issue it is very likely that in uraemic patients myocardial ischaemia tolerance is markedly reduced even in the absence of classical arteriosclerosis due to structural and metabolic abnormalities of the myocardium. Furthermore endocrine and metabolic disturbances such as secondary hyperparathyroidism and prolonged anaemia are unique contributors to cardiovascular changes in renal failure patients. Other hormonal changes, that have been described as playing a role in renal disease progression, such as an activated renin angiotensin system are now increasingly recognized as playing a role in generalized arteriosclerosis as summarized by Lottermoser, Vetter and Düsing. Krane and Wanner deal with the fact that the uraemic patient accumulates many of the well and less well described cardiovascular risk factors, even if their interpretation sometimes is difficult as in the case of serum cholesterol levels, where a J-shaped curve phenomenon can be seen. The same phenomenon is described in Rigatto's and Parfrey's article regarding blood pressure. Finally, the best way to replace renal function in order to treat and prevent progression of heart disease remains to be defined. As stated by Lameire and Hoeben, many more studies will be needed to define optimum care for these high risk patients.

All these observations make it clear that papers like the ones in this issue are necessary to improve our understanding and awareness of this increasing health care problem.

References:

1. Schmidt A, Stefenelli T, Mayer G. Informational contribution of non invasive screening tests for coronary artery disease in patients won chronic renal replacement therapy. Am J Kidney Dis 2001 37: 56–63.

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